and the prognosis for resolution of acute attack is excellent.⁴

Acute crystal arthritis (acute gout or pseudogout) of a prosthetic joint is a relatively rare occurrence, and, as such, only a few cases have been reported.⁷⁻¹¹ Although it is of prime importance to exclude implant sepsis, diagnosis of acute crystal arthritis must be excluded as this condition can be treated by simple pharmacological means. Preceding episodes of gout or pseudogout or predisposing medical conditions should raise the possibility of crystal induced arthritis. Physical examination should include a detailed survey to exclude other involved joints. Immediate joint aspiration is indicated in all cases of suspected infected joint arthroplasty and should be among the first diagnostic tests done.^{12 13 14} The aspirate should routinely be sent for Gram stain analysis; bacterial and fungal cultures; white cell count and differential; and biochemical and crystal analyses.¹⁵ Synovial fluid crystal analysis, Gram stain, and appropriate cultures are the only reliable means of distinguishing between the acute crystal and septic arthritis. Failure or delay in recognising acute gout or pseudogout can lead to inappropriate treatments, such as the unnecessary use of antibiotics, surgical arthrotomy, or removal of the implant. Acute crystal arthritis should be excluded by polarising light microscopy of synovial fluid aspirate before removing implant components.

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Pyoderma gangrenosum in ulcerative colitis: considerations for an early diagnosis

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Pyoderma gangrenosum is a poorly understood destructive cutaneous disorder, characterised by progressive painful ulceration.¹ Accurate epidemiological data are missing, but in half of cases there is an associated underlying disease, most commonly inflammatory bowel disease, rheumatological and haematological disorders.^{2 3} As pyoderma gangrenosum is not commonly encountered by clinicians, the diagnosis of such lesions is not always straightforward. We report the case of a man with ulcerative colitis, who presented with a non-healing ulcer of traumatic origin unresponsive to conservative and surgical management. This case emphasises the importance of detailed history taking and the consideration of pyoderma gangrenosum as a differential of such lesions in patients with a background of related systemic disease.

Case report

A 49 year old man was referred to the department of plastic surgery with a two week history of a post-traumatic, painful, and expanding ulcerated area on the anteromedial aspect of the left lower limb. The patient had a past medical history of ulcerative colitis,

which he reported to be in remission for several years and he was currently not on any medication, including steroids. On initial presentation, the lesion was well demarcated measuring 6 × 4 cm with slightly undermined edges. Examination was otherwise unremarkable and conservative management was initiated. A week later, the lesion expanded to 12 × 8 cm, after which we debrided the wound and covered it with a split thickness skin graft. The postoperative course was uneventful, and the patient was discharged.

Six months later, the patient returned with a new ulcerated lesion, adjacent to the previous skin graft. Again, it was reported as post-traumatic. The lesion measured 5 × 8 cm (figure) and was indurated with violaceous undermined edges, atypical of a traumatic ulcer. Cultures were sterile and blood tests showed a normocytic anaemia and raised erythrocyte sedimentation rate.

Cytological examination of scrapings from the ulcer base showed inflammatory features, and there was no bony involvement on radiography. The atypical features of the lesion and the abnormality of the blood tests prompted us to revisit the history. In depth questioning found that the ulcerative colitis was not inactive

Appropriate evaluation and critical interpretation of findings are essential for early diagnoses of pyoderma gangrenosum

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Ulcerated lesion measuring 5 × 8 cm, on the middle third of the anterolateral aspect of the lower left leg, showing well demarcated violaceous edges

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as reported and the patient was in fact experiencing bleeding from the rectum and had neglected to take his recently prescribed mesalazine and prednisolone.

Based on these findings, we considered the possibility of pyoderma gangrenosum as a differential, and we took an incisional biopsy of the lesion. Histopathology showed oedema, lymphocytic vasculitis, and neutrophilic infiltrates—features consistent with pyoderma gangrenosum.

The patient promptly started a course of 40 mg of oral prednisolone once daily and topical clobetasol. After a month, the ulcer had almost completely healed. In retrospect, the initial rapidly expanding ulcer that was treated with a skin graft was probably undiagnosed pyoderma gangrenosum.

Discussion

The diagnosis of pyoderma gangrenosum is based primarily on clinical presentation and course; histology can also be of value. The causes of pyoderma gangrenosum are unknown, but autoimmune mechanisms are probably implicated.⁴ Inflammatory bowel disease is the most common underlying disorder and is found in 10–15% of pyoderma gangrenosum cases.⁵ Of those with active ulcerative colitis, 7.1% may develop ulcerative colitis as an extraintestinal manifestation.⁶ Occurrence at sites of trauma or surgery is rare but well documented.⁷

Pyoderma gangrenosum most commonly occurs in adults aged 30–50, on the lower extremities and trunk. It usually begins as tender papulopustules that break down to form expanding ulcers with raised, violaceous, and well demarcated edges.¹ Histopathology of pyoderma gangrenosum is non-specific but can help to differentiate it from lesions with similar features, most commonly infective, neoplastic, and rheumatological associated ulcers.^{4, 8}

Systemic steroid therapy remains the treatment of choice and leads to rapid relief of pain and initiation of healing in most patients. Infection must be excluded before starting immunosuppressive treatment.⁹ Successful treatment of the underlying disease results in complete remission or dramatic improvement of pyoderma gangrenosum.

Our case shows the importance of careful history taking and critical interpretation of individual findings

for an accurate diagnosis and thus appropriate management. Although a retrospective diagnosis of pyoderma gangrenosum seems obvious, the report of quiescent ulcerative colitis and a traumatic cause of the ulcer was misleading. Had the non-healing traumatic ulcer been associated with a predisposing systemic disease, the diagnosis of pyoderma gangrenosum may have been reached sooner.

In fact, surgical debridement of ulcers should not be performed without confirmation of the underlying cause, as in patients with pyoderma gangrenosum it may cause extension of the necrotic area and even precipitate pyoderma gangrenosum at the donor site.⁷

This case emphasises the importance of connecting findings from history, examination, and investigations. The finding of anaemia should have led to the suspicion of active ulcerative colitis or at least prompted investigation as to its cause. The case also highlights that a patient's account of the history cannot always be taken at face value and, on occasion, further probing may be necessary. Finally, the case stresses that pyoderma gangrenosum should be considered in non-healing rapidly expanding ulcers, particularly in patients with a background of related systemic disease, even with a traumatic cause.

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Endpiece

Medical people in Paris

I have no faith in the medical people here; not one of them seems honest to begin with. To get patients and to humour them when got seems much more the object of these people than to cure their ailments. In fact, what can they know about one's ailments allowing only some three minutes to the most complicated cases! And so I leave my case to nature; and nature seems to want either the will or the power to remedy it.

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